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CONTINUOUS SPINAL ANESTHESIA IN ABDOMINAL AND THORACIC SURGERY*

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EXPERIMENTATION

In 1938 animal experimentation was made to establish the safety and controllability of continuous spinal anesthesia. It was found that by permitting the spinal puncture needle to remain in the subarachnoid space of animals, and at regular intervals adding small doses of the anesthetic drug, the anesthesia could be kept up for as many hours as desired. It was also found that animals could take, with safety, many times the lethal dose of the drug if it was administered in small doses over a long period of time. It was established that small doses of a drug given over a period of time was much safer than one large single dose. Dogs and monkeys were used, and novacaine (procaine hydrochloride) was the drug used. Novocaine is accepted as the safest drug, clinically and experimentally, for producing spinal anesthesia.

We kept monkeys under continuous spinal anesthesia for as long as twelve hours, and used more than 600 mgs. of novocaine, injecting 50 mgs. at thirty minute intervals. The height and degree of anesthesia was controlled as desired. Some monkeys had a total of more than fifty hours of continuous spinal anesthesia without any demonstrable deleterious effects.

CLINICAL OBSERVATIONS

Clinically, the most important disadvantages of the single-dose method of spinal anesthesia are: (1) failure of the drug to "take" or produce anesthesia to the desired level or degree; (2) the action of the drug wearing off before the operation is completed; and (3) toxic symptoms or even sudden death following the intraspinal injection of a large single dose of the anesthetic agent. We felt that our animal experimentation with this new method of continuous spinal anesthesia had eliminated these disadvantages found in the single dose spinal anesthesia method.

On April 10, 1939, we gave our first continuous spinal anesthesia to a patient. This patient was jaundiced due to stones obstructing the common bile duct. She also had fever, chills, and sweats. There was present an acute cholecystitis. After proper preoperative preparation with improvement in liver function, she was operated upon using continuous spinal anesthesia. The initial dose of novocaine was 200 mgs. which produced anesthesia to the fourth thoracic segment in approximately 10 minutes. There was perfect abdominal relaxation. The stones were removed from the common bile duct, and a T-tube was placed for drainage. The gall bladder was removed. The intestines began to distend, and the abdominal muscles began to get tense when the peritoneum was grasped for abdominal closure, 35 minutes after the initial injection of novocaine. The stopcock was opened and 20 mgs. of novocaine was given. The intestines promptly collapsed, and the abdominal muscles relaxed within two minutes after the injection of novocaine. The abdominal incision was satisfactorily closed in a few minutes under ideal operating conditions. Experienced abdominal surgeons are familiar with the difficulties encountered in closing upper abdominal incisions in patients with unrelaxed, tense, or rigid abdominal muscles. Indeed, the wound closure may be more trying and difficult than the operation itself. With continuous spinal anesthesia perfect muscular relaxation may be maintained at all times during the operation. This is a source of comfort to the surgeon and patient. We have now used this anesthesia in more than 4,000 cases. As our experience has increased, we have extended the field of usefulness of this type of anesthesia. It was first used in abdominal, pelvic, perineal, and

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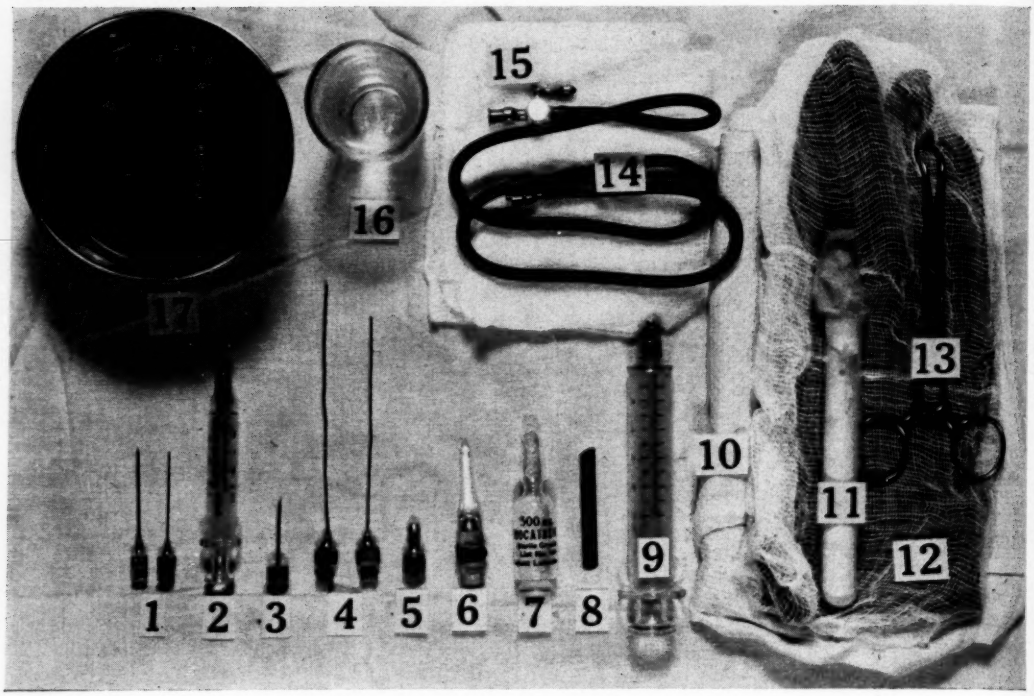


lower extremity operations. We now use it in thoracic, breast, and upper extremity operations. It was first limited to the middle age group of patients, but now we use it on any age patient if there are no other contraindications. In breast amputations there is less bleeding, less renal and respiratory irritation, and a much more rapid convalescence than when general anesthesia is used. We have found it to be safe and easily controlled in breast operations. Difficulties with this method of spinal anesthesia are usually due to improper spinal puncture or insufficient dosage of the drug. Difficulties diminish or disappear as experience increases in the use of this method of anesthesia.

EQUIPMENT AND TECHNIQUE

Figure I

1. Indicates hypodermic needles.
2. A 2 cc. syringe with needle attached.
3. A Sise introducer used to puncture skin.
4. 18 and 19 gauge malleable needles.
5. Luer-Lok plug.
6. Ampoule containing 50 mgs. ephedrine sulfate in 1 cc. of 2% novocaine.
7. 500 mg. ampoule of novocaine crystals.
8. File used to open ampoules.
9. A 10 cc. Luer-Lok syringe.
10. Sterile towel.
11. Sterile test tube of powder.
12. Sterile rubber gloves.
13. Towel clip.
14. 30 inches of small caliber, thick walled rubber tubing.
15. Stopcock.
16. Medicine glass filled with sterile water.
17. Metal container filled with fluid used in preparing the skin for puncture.

Figure II

4. Illustrates the head end of a mattress that is 6 feet long, 18 inches wide and 5 inches in height. This mattress is covered by rubber.
5. Shows a buckle and strap which is used in removing the lower end of the mattress when perineal operations are done.
   (a) Shows cut out portion of mattress for perineal operations.
6, 7, & 8. Lower end of mattress used to support the lower extremities. The breaks conform to the legs and thighs when flexed and the patient is in the Trendelenburg position.
9. Insert illustrates the opening in the pad enlarged so that the needle may be adjusted if necessary.
10. Illustrates a 30 cc. Luer-Lok syringe with 30 inches of rubber tubing and needle attached.
11. Illustrates a stopcock.
12. Shows a short bevel malleable spinal needle with an opening in the bevel. This opening facilitates the flow of spinal fluid, and prevents blockage to the flow by the dural membrane.
13. Illustrates a Luer-Lok connected to a malleable spinal needle.
14. Luer-Lok plug used to prevent flow of spinal fluid while solutions are being prepared.
15. Sise introducer used to puncture the skin so that the malleable spinal needle may be introduced without difficulty.

Figure III illustrates some of the positions that patients are placed for operation.

C. (1) Position for operations on the thorax and breast operations.
(2) Position for operations on the abdomen and pelvis.
D. (3) Position for paravertebral thoracoplasty or other operative procedures on the thorax.
(4) Position for kidney operations or sympathectomies.
E. (5) Position for sacral operations, pilonidal sinus, and operations on posterior surface of extremities.

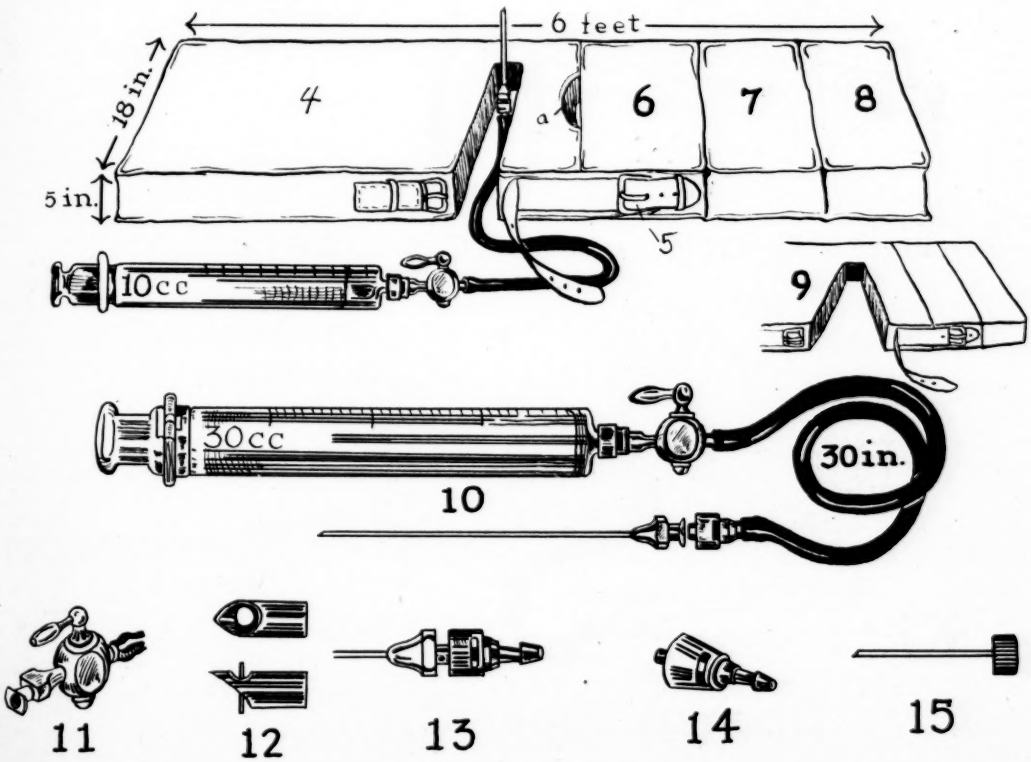
Figure IV

A. Illustrates the patient in position for plastic and perineal operations.
B. Trendelenburg position for pelvic, abdominal and thoracic operations.

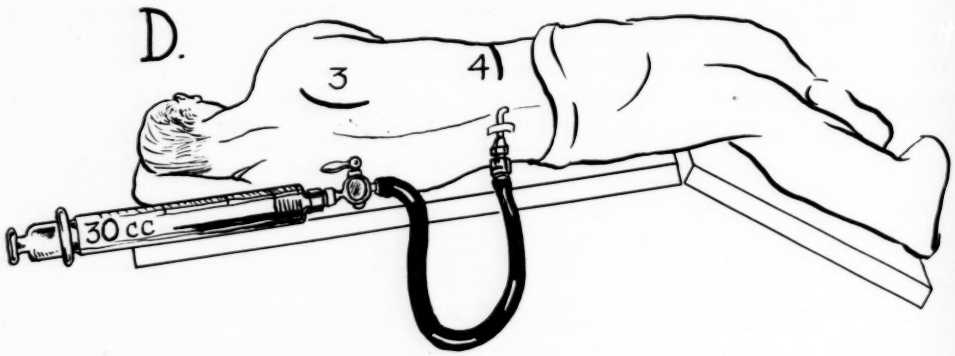
ADMINISTRATION

Preoperative Medication—we believe the preoperative medication to be very important in continuous spinal anesthesia. The average healthy adult gets 3 grains nembutal at eight o'clock the evening before operation. This insures a good night's rest, and diminishes nervous apprehension. Three hours before operation, 3 grains of nembutal is given. One hour before operation a hypodermic injection is given containing 1/4 grain of morphine sulfate, and 1/100 grain of scopolamine hydrobromide. These doses are varied according to the individual patient. In bad risk, elderly patients the dose is diminished. We do not increase the above dosage. If patients are not sufficiently sedated, 1/8 grain of morphine sulfate is given intravenously, and is repeated as often as necessary.

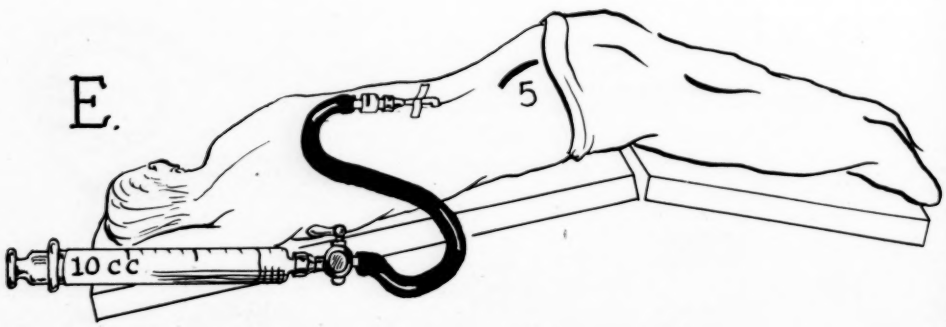
The patient is placed on a specially designed mattress, Figure II (4, 6, & 8). The operating table, with mattress and patient, is placed in 3 degrees Trendelenburg position. The patient is turned on one side with back towards the opening in the mattress, and thighs are flexed on abdomen. This increases the space between the lumbar spinous processes, and facilitates successful lumbar puncture. The third, fourth, or fifth lumbar interspace is chosen, and the skin over the chosen interspace is infiltrated with an intradermal injection containing novocaine 2% and ephedrine. The ampoule contains 1 cc. of 2% novocaine and 50 mgs. of ephedrine, and after the skin is infiltrated the remainder is injected hypodermically and into the interspinous ligament. The skin is now punctured



Abdominal and
Thoracic Operations

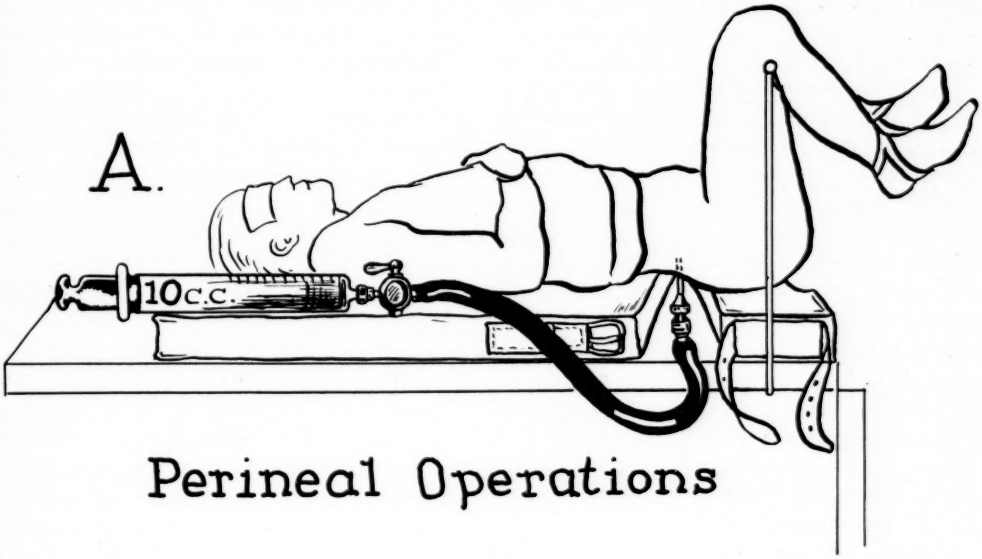


Kidney and Thoracic Operations

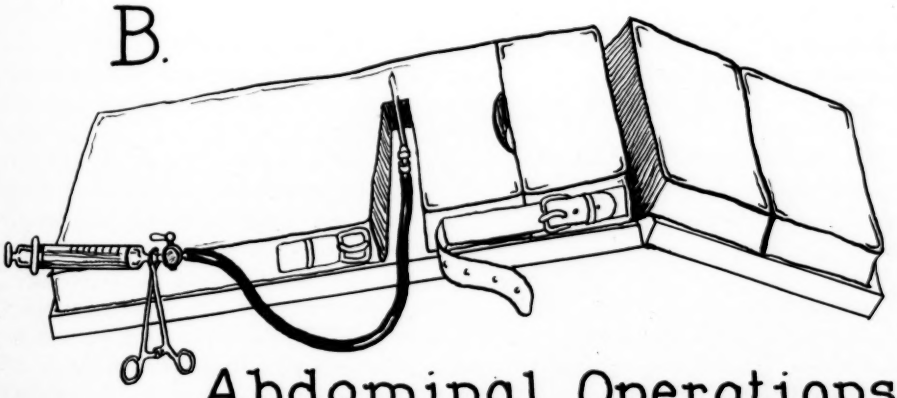


Sacral Operations

using a Sise introducer, Figure II (15). A malleable needle, No. 18 or 19 gauge, Figure I (4), is now introduced into the subarachnoid space and a free flow of spinal fluid obtained. A stilette is not used in the needle. Ten cc. of spinal fluid is removed by attaching a 10 cc. Luer-Lok syringe to the needle in the subarachnoid space, Figure I (9). The syringe is detached, and the flow of spinal fluid is controlled by attaching a Luer-Lok plug, Figure II (14). A needle is attached to the 10 cc. syringe containing 10 cc. of spinal fluid and this fluid is thoroughly mixed with the novocaine crystals in the 500 mg. ampoule, Figure I (7). The file, Figure I (8), is used in opening the ampoules, Figure I (6 & 7). The dissolved crystals are now removed from the ampoule into the syringe which is attached to 30 inches of rubber tubing, Figure I (14). It takes 2 cc. of this solution to completely fill this rubber tubing. The stopcock, which is connected with the syringe, is now turned to



Perineal Operations



Abdominal Operations

Trendelenburg Position

cut off the flow of solution. There will be 8 cc. of solution remaining in the syringe and 2 cc. in the 30 inches of rubber tubing. The plug is now removed from the needle in the spine, and the rubber tubing, with attached syringe, is connected with the spinal needle. The stopcock is turned to open, and by gentle traction on the plunger of the syringe a free flow of spinal fluid is obtained. Sometimes the spinal fluid flows itself, pushing the plunger of the syringe outward. When we dissolve 500 mgs. of novocaine in 10 cc. of spinal fluid we get a 5% solution, each cc. of which contains 50 mgs. of novocaine. The usual initial dose of novocaine is 150 mgs.; 3 cc. of this solution is slowly injected. The desired height and degree of anesthesia should be obtained in five or ten minutes. The patient is now placed into the position desired for operation. As soon as that position is assumed, the flow of spinal fluid is tested to be sure it is free. The operative area is prepared, draped, and the operation begun. Every thirty minutes 1 cc. of spinal fluid is aspirated to be sure of a free flow, and 2 cc. returned to the subarachnoid space. This gives a dose of 1 cc. containing 50 mgs. of novocaine. Sometimes the novocaine must be given more often than 30 minute intervals, and in doses more than 50 mgs. in order to maintain perfect anesthesia. The proper dose is enough to produce the desired level and degree of anesthesia, and this varies greatly.

SUMMARY

1. Continuous spinal anesthesia has been used in more than 4,000 surgical operations.
2. The technique of producing continuous spinal anesthesia is discussed.
3. The safety and controllability of this method is presented.
4. We believe that our mortality and morbidity have been lowered by the use of this method of anesthesia.

DISCUSSION

DR. R. DOUGLAS SANDERS (Wilmington): I welcome the opportunity of addressing this Society and of meeting with you and particularly of meeting Dr. Lemmon whom I have heard so much about for so long and have used his technique for quite a while.

It is possible that the conditions that exist at Jefferson Medical College, where no professor of anesthesia is in residence, established the need for an improvement in spinal anesthesia, and I want you to clearly understand that my remarks shall not in any way detract from the advantage that has been gained for medicine by Dr. Lemmon's singular work on this particular technique. It has been of distinct advantage to us and has enlarged the field over which spinal anesthesia might be used to good advantage.

Certainly spinal anesthesia may be given over a much more prolonged period and over a greater group of serious risk cases than formerly was possible. We have not, however, been able to use it in those cases which we regard as absolutely inflexible contra-indications; to mention one, any degeneration of the spinal cord such as occurs in pernicious anemia, diabetes or late neurosyphilis.

Too, we do not use it in cases of extreme shock even in smaller doses. We have found that inhalation is preferable. You will understand my remarks are only my experience and are not based on others' work. As for novocaine selectively attacking the sensory system without concomitantly attacking the sympathetic, it seems to occasionally do so in the very aged who have a very low threshold of effect of procaine. In the heart is very much slower because all the mechanisms of the heart have been paralyzed. I think an absolutely minimum lethal dose of novocaine does not exist, that truly thousands of milligrams may be given. The reason for the development of such serious shock in cases that have had the one shot method is frequently the paralysis of the sympathetic system suddenly. One hundred and fifty milligrams is a very small dose to inject subcutaneously but a very high dose to inject intraspinally if you inject it rapidly in a patient who is a serious risk. In this type of instance the continuous method would have been proven very valuable and to all extent it is reversible early. Later on we have not been able to reverse it completely.

As to the relative safety of the drugs that may be used in the continuous method we still hold that novocaine is the safest known local anesthetic and we prefer it, although many are now using pontocaine and the new bricaine. We still stock with novocaine. We

find that the dosage tends to be higher with the continuous method than with the one shot method for the reason we begin more cautiously and we stop all the pain we may. We are not limited by the fact that we cannot again give another dose. Of course, by various other methods that have recently come into vogue it has been possible to produce a spinal-like relaxation. By the use of socopropaine we have produced a relaxation possibly even greater than spinal, if that is conceivable, but we do not like to use them where spinal can be used, and we rather tend to limit our use of even continuous spinal to abdominal operations for the reason we wish absolute control of the respiratory mechanism.

Again let me thank you gentlemen for the privilege of addressing you and again I appreciate having met Dr. Lemmon.

DR. ROBERT S. HELLER (Wilmington): I have little to add to what has already been said. One of the faults we found with continuous spinal, particularly in working around the stomach, has been the nausea, not exactly the pain but the discomfort that is associated with it. We have had quite frequently to supplement the continuous spinal with penthothal and, of course, once we start using that we have to continue it all during the operation. We have used pontocaine and glucose in place of novocaine. After having used both types of agents, we found that in using pontocaine we were able to use a much, much smaller dose by comparison than we would with novocaine.

I don't think I have anything else to add to that. Thank you.

DR. LEMMON: I want to thank the members of this society for the privilege of being here to give this talk. Dr. Sanders' and Dr. Heller's discussions were very valuable and are much appreciated. If I had Dr. Sanders or Dr. Heller available for giving anesthesia I would depend upon them completely, so that I would be free to do the operations without being troubled with the anesthesia. At the present time we do not have a physician anesthetist available and I have developed a type of spinal anesthesia that is under direct control.

I agree with Dr. Sanders that degeneration of the spinal cord, neurosyphilis, and shock are contraindications to the use of spinal anesthesia. In pernicious anemia there are degenerative changes in the spinal cord due to the anemia. If spinal anesthesia is used on these patients it may get the credit for having produced the degenerative changes in the spinal cord. As far as shock is concerned I don't think any patient should be operated on in shock unless it is controlled or being controlled while anesthesia is being used. If it is due to hemorrhage, multiple transfusions will soon relieve the condition and anesthesia and operation may proceed with safety. This is particularly true in cases of severe hemorrhage due to peptic ulcer.

Some anesthetists have more experience with pontocaine than with procaine. There is no argument about the drugs to be used. The anesthetist should use the drug with which he has had the most experience.

This method of anesthesia has been found to be safer than the original one shot dosage of spinal anesthesia. We have found that spinal anesthesia is the best anesthesia for many patients. We like to end with the anesthesia that we began with, particularly when it is the anesthesia of choice. This method of anesthesia will last regardless of the duration of the operation. We feel sure that we have reduced both mortality and morbidity in surgery by the use of this method. Other surgeons have used it for a variety of conditions and they have made improvements in its use. I am always pleased to hear of these improvements.

RHEUMATIC CARDITIS WITH CONGESTIVE FAILURE

Presentation of a Case

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A fourteen-year-old girl entered the Kent General Hospital because of heaviness in chest, soreness in both elbows, and fever.

The patient was well until a month prior to admission when she "caught cold," had sore throat, fever, and generalized abdominal pains which lasted several days, was well for a week then had recurrence of abdominal pains and slight chest pains. She recovered

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from this episode after several days and attended school for about ten days when she became ill with a fever of 101, backache, a sensation of heaviness in chest, soreness in both elbows, and mild abdominal pains. Several days after the onset of these symptoms she was admitted to the hospital.

Past history revealed measles, mumps, pertussis, and chickenpox in childhood. She had infrequent colds and earaches occasionally as a child and mild autumnal hay fever. Tonsillectomy at age nine. At age ten she had acute sore throat followed by migratory polyarthritis, subcutaneous nodules, and high fever. A diagnosis of rheumatic fever was made at that time, and the patient was confined to bed for six months. During the six months following this illness, she had what her mother described as "St. Vitus Dance," and the family was advised that she had a heart murmur. Her menses began at age thirteen and were normal.

Systemic review revealed epistaxis two weeks before admission, mild anorexia since onset of illness, no nausea or vomiting, slight cough, pain in chest, and choking sensation on deep breathing—of several days duration. No true dyspnea and no ankle edema. Usual weight—123 lbs.

Family History. A maternal aunt had rheumatic heart disease.

Physical examination revealed a sandy-haired, fourteen-year-old girl of fair complexion, fairly well developed, well nourished and in no acute distress. Temperature, 102; pulse, 100; and respirations, 22. Eyes—conjunctiva normal, fundi, negative. Ears—negative. Nose—negative. Throat—negative, except for slight post pharyngeal phlegm. Mouth—negative. Neck—negative. Lungs—resonant to percussion, no rales, breath sounds normal, expand equally. Heart—apical impulse forceful four inches to left of midline, not enlarged to percussion but increased dullness over lower end of sternum, P2 markedly accentuated, crescendo type presystolic and early diastolic murmurs heard transmitted to left axilla. Abdomen soft, no localized tenderness, no abnormal masses. Liver and spleen not palpable. Skin—no petechiae found. Pinpoint hemorrhagic lesions were scattered over both legs which she thought were due to flea bites from her dog who slept with her. Extremities—no clubbing of fingers, elbows not tender or swollen, pain occurred on active and passive motion at elbow joints.

Admission urinalysis—ph 7.0, amber color, sp. gr. 1.020. Albumin negative. Sugar negative. Microscopic — occasional RBC and WBC. Blood count showed RBC—3,160,000, Hemoglobin—75%, WBC—10,300, Differential: polys—84%, lymphocytes—3%, and monocytes—14%. Sedimentation index was 27 mm. in 60 min. with vertical type curve.

A positive diagnosis of rheumatic heart disease with mitral stenosis was made, and the present illness was thought to be due to a recurrence of acute rheumatic fever or subacute bacterial endocarditis.

On admission blood culture and blood cultures on three consecutive days thereafter all showed no growth. Chest xray revealed heart enlarged in all diameters and basal passive congestion. Electrocardiogram showed normal sinus rhythm, rate 125, notching of P waves in leads one and two, P-R interval 0.20 and right axis deviation—findings suggestive of a rheumatic process.

For eight days after admission the patient received penicillin, 50,000 units q. 3 h. with no effect on her fever. Temperature continued to range from 100 to 102 during this period. However, patient had no complaints except a sensation of heaviness in the chest. Repeated observations revealed no evidence of petechiae of skin or mucosa. About one week after admission on cardiac auscultation, a faint rough sound of unusual character with each systole was heard in the fourth interspace to left of sternum, and it was thought that a pericarditis of rheumatic origin might be developing. In the absence of any positive findings of subacute bacterial endocarditis a tentative diagnosis of active rheumatic myocarditis and rheumatic pericarditis was made; penicillin was discontinued and patient treated with sodium salicylate gr. xxx q. 4 h. The temperature dropped to 98.8, but after one day's treatment she developed marked tinnitus. Salicylate was discontinued. The next day she had a slight erythematous, macular, patchy rash about her neck, trunk, forearms, and knees. A final sedimentation index was 19 mm. in 60 min. Eleven days after

admission the patient was transferred "unimproved" to a university hospital for further study and treatment.

At the university hospital on admission her temperature was 101.6, pulse 120, respirations 24, blood pressure 105/60 and positive findings on physical examination were—weight 107 lbs. A fairly well developed, well nourished, white female of fourteen who appeared chronically ill, with moderate pallor of the skin and a light malar flush. There was a splinter hemorrhage under the third right fingernail, no petechiae, no distention of cervical veins. Cardiac examination—heart overactive and enlarged to both right and left. Fullness in the region of the pulmonic conus. Diastolic thrill at apex and a diastolic tap in the pulmonic area. On auscultation, the first mitral sound was snapping and preceded by a crescendo diastolic murmur and followed by a blowing, systolic murmur. The second pulmonic sound was accentuated. To the left of the sternal border in the third and fourth interspaces, there was a blowing diastolic murmur continuous with the second sound. Abdomen—flat, liver one f. b. below the c. m. Rectal examination normal, no clubbing of fingers or toes, no arthritis, neurological examination normal.

Laboratory data: STS, negative. RBC 4.2 million. Hgb. 11 gms. Ht. 35. Sed. rate, 57/34. WBC 8,000. Differential normal. Urine: amber, clear, sp. gr. 1.030, acid, sugar negative, albumin negative; microscopic, 4-5 WBC per hpf. Stool examination normal. No occult blood. NPN, 20-48 mgm.%. Chloride, 89-104 m. eq. per liter. CO2 combining power, 23-30 m. eq. per liter. Fasting sugar, 94 mgm.%. Cholesterol, 167 mgm.%. Bilirubin 1.4 mgm.%. Total serum protein, 6.3 to 7.7 gms.%. Albumin 4.0 to 4.8 gms.%. Globulin, 2.3 to 3.9 gms.%. Alkaline phosphatase activity, 3.5 units. Urea clearance, 75% normal maximum clearance. PSP. 62-65% excretion in two hours. Salicylate level, 97-204 gamma per cc. Venous pressure, 104-135 mm. of saline. Circulation time, 18 seconds, decholin method. Vital capacity, 1200 cc. EKG on admission: Normal sinus rhythm. PR interval .12 seconds. Right axis deviation. P1 notched. Tracing compatible with rheumatic heart disease. Blood cultures: Thirty-six blood cultures taken. Thirty-two cultures sterile. Four different organisms were obtained from the remaining four cultures, all probable contaminants. Cultures were grown aerobically and anaerobically under carbon dioxide and in several media. Throat culture: Normal throat flora. Cephalin flocculation positive, 3 plus. Tuberculin PPD, first test dose, negative. Xray of chest on admission showed heart considerably enlarged, triangular in outline with fullness in the region of the pulmonic conus and some congestive changes in the lung fields.

Course. The patient appeared acutely and chronically ill on admission to the hospital. She was thought at that time to be suffering from active rheumatic pancarditis. The possibility that she might also have subacute bacterial endocarditis was also entertained. She was placed on bed rest and treated with aspirin, four gms. daily, which she tolerated only fairly well. Despite salicylate therapy, her temperature rose on one occasion to 103.2 during the two week period in which she received this medication. Ten days after the initiation of aspirin therapy, the patient developed an urticarial eruption together with nausea and tinnitus. Because of her apparent sensitivity to aspirin, this drug was discontinued two weeks after it was started. At this time, her temperature averaged about 99.6 and she had moderate tachycardia; she also developed several new splinter hemorrhages beneath her nails. Following the discontinuance of salicylates, her temperature rose slightly.

Because of a fall in hemoglobin from 11 to 8 gms., the patient received a transfusion of 500 cc. of blood three weeks after admission, and again four days later. Since it was considered possible that the patient had subacute bacterial endocarditis, penicillin therapy with six million units daily by i. m. injection was begun. At this time, she had developed slight clubbing of her fingers. Penicillin was continued for seven days, a total of 43,500,000 units being administered. There was no significant effect upon her temperature. Penicillin therapy was begun again two weeks after her first course and during the next week she received 67,000,000 units of penicillin. Five days after initiation of second

penicillin course by continuous intravenous drip, the patient went into acute pulmonary edema and was digitalized with lanatoside C. This measure, coupled with the discontinuance of the i. v. infusion was followed by a prompt restoration of cardiac compensation. Interestingly, the patient's temperature which had been rising to 102 or 103 prior to the discontinuance of penicillin, fell to 100 after the chemo-therapeutic agent was stopped.

During the next week, her temperature remained essentially unchanged following which it began gradually to rise to levels fluctuating between 101 and 103. Despite persistently negative blood cultures, it was decided once again to attempt penicillin therapy and three weeks after last course (about the second month of hospitalization) the constant infusion of 25,000,000 units of penicillin daily in 5% glucose solution was begun. On the day after this was begun she began to have auricular fibrillation confirmed by EKG and became acutely ill. Her liver increased in size; she developed nausea and vomited. During the next two days, quinidine was administered in an unsuccessful attempt to restore the patient's cardiac rhythm to normal. Penicillin therapy was discontinued after the administration of 100,000,000 units in this course. Five days later the patient's cardiac rhythm reverted spontaneously to sinus rhythm. At this time, it was observed that the patient's Hgb. had fallen to 7.5 gms.

Because of her poor cardiac reserve, she was transfused on two occasions with washed red cells suspended in 5% glucose. Following these transfusions, her hemoglobin rose to 11 gms. and remained at this value until her discharge from the hospital. Throughout her stay on the ward, it was uncertain whether this patient had subacute bacterial endocarditis. Although she had several crops of splinter hemorrhages and occasional petechiae in the buccal mucosa along the line of dental closure, at no time did she have hemorrhages in her fundi, or hematuria. On several occasions, the tip of her spleen was thought to be palpable, but the presence of definite splenic enlargement could never be determined with certainty. Her WBC fluctuated between 7,000 and 11,000.

After an interval of three or four days following her last penicillin treatment, treatment with amidopyrine was begun following which the patient's temperature fell promptly and during the next week showed diurnal variations between 97.6 and 99.8. The patient tolerated this drug with some difficulty, and its use was discontinued for a short period following which her temperature rose to 101. Following the readministration of amidopyrine, her temperature fell again to levels slightly above normal. In the week prior to discharge an EKG revealed sinus tachycardia. PR interval 0.16 seconds. T waves inverted in leads 2 and 3. Tracing shows digitalis effect. At this time the patient developed a high pitched musical component to the systolic murmur which had been audible previously in the mitral and apical areas. This murmur had the characteristics associated usually with active rheumatic fever. At no time during her stay in the hospital did the patient have joint symptoms. Because of her relatively static condition and her intense desire to return to her home, she was allowed to leave the hospital.

Clinical Impression. Rheumatic heart disease, active, with mitral stenosis and insufficiency, aortic insufficiency, cardiac enlargement and failure. Paroxysmal auricular fibrillation and subacute bacterial endocarditis. Sensitivity to salicylates.

The patient was discharged three months after admission, unimproved, with instructions to remain at complete bed rest at home, to take a cardiac normal 4 gm. salt diet, and digitalis 0.1 gm. five days a week, also amidopyrine 0.3 gm. twice daily, and to seek the care of her family physician.

The patient got along fairly well for about a month after her return from the university hospital, but in spite of careful treatment for congestive heart failure, gradually developed increasing dependent edema, dyspnea and orthopnea. Adequate digitalization was insufficient to maintain her in a compensated condition, and recourse to mercurial diuretics was taken. She received frequent injections of 1 cc. salyrgan and initially these were followed by prompt diuresis and reduction in edema, but after repeated injections she developed oliguria and failed to show any improvement. Her temperature continued to be

elevated somewhat until after about two months at home when it became subnormal and persisted at subnormal levels until the last examination recorded.

At the last examination, two and a half months after her discharge from the university hospital and about seven months after the onset of her symptoms, the patient was orthopneic, pale and emaciated in appearance. Temperature, subnormal, 95, pulse 112, MSR—but thready. Respirations 36, shallow and difficult, no petechiae of skin, mucous membranes, or fundi. Lungs—coarse, bubbling rales at both bases. Heart—murmurs as previously described but apical impulse prominent in the 6th interspace, anterior axillary line, and demonstrable enlargement to percussion—both of left ventricular border and to right of sternum, prominent venous pulsations in neck veins and distention. Abdomen—liver, two f. b. below cm., marked distention of abdomen with dullness to percussion and fluid wave, pitting edema of sacrum and trunk posteriorly as high as costovertebral angles and four plus pitting edema of both lower extremities.

Final Clinical Diagnosis. Rheumatic Heart Disease, Active Congestive Heart Failure, Severe.

Autopsy. Several days following the last recorded examination, the patient died and autopsy was performed. The body which retained some antimortem heat was that of a young girl. There was edema of the sacral region, genitalia, and lower extremities. On opening the chest a small amount of free fluid was found in both pleural cavities, and thin pleural adhesions, bilaterally. There were some dense, tough, diaphragmatic adhesions on the left. Over the anterior surface of the pericardium was a heavy network of blood vessels. Pericardium was thick and everywhere adherent to the epicardium with fibrinous adhesions. There was no free pericardial fluid. Heart weighed 610 gms.; it was enlarged and the chambers dilated. Myocardium of right ventricle was two times normal thickness while that of left ventricle was one and one half times normal thickness. Papillary muscles hypertrophied and columnae carneae prominent, the endocardium over about one-third of surface of left atrium was irregularly roughened with dull red patches. Tricuspid valve leaflets were slightly thickened and fused; a row of minute gray vegetation extended along the entire contact edge. The pulmonary valve appeared normal. Mitral valve leaflets likewise thickened, fused and roughened along the contact edge with a row of minute, slightly red vegetations. There was a similar thickening of aortic leaflets with vegetation along the contact edge and slight fusion of commissures. There was stenosis and probable regurgitation of the tricuspid, mitral, and aortic valves.

The peritoneal cavity contained several liters of straw-colored fluid; liver was somewhat enlarged and smooth with prominent markings; spleen small and firm.

Microscopic examination of sections from heart, pericardium, spleen, liver and kidney were reported by pathologist* as follows:

Heart: The myocardial cells are enlarged, and the fibers are separated by edema fluid. The pericardium is covered with a layer of granulation tissue through which there is a slight infiltration of lymphocytes. The aortic valve is irregularly thickened with a mass of hyaline connective tissue. Small blood vessels extend out into the thickened portion and are accompanied by a few lymphocytes. There is a small projection from the thickened surface. This consists of the same connective tissue, but it is edematous. The surface of the mass is covered with a smooth thin layer of endothelium, and there is nothing to suggest bacterial endocarditis. Careful search through the heart sections shows no evidence of the so-called "Aschoff's bodies."

Pericardium: A section of the parietal pericardium is covered with young scar tissue and granulation tissue through which there are numerous lymphocytes and monocytes.

Spleen: The organ is markedly congested, but shows no other pathology.

Liver: There is a marked degree of passive congestion characterized by distension of the sinusoids around the hepatic veins. The distension is so great that the adjacent liver cells are frequently squeezed out of existence. The congestion extends over half way toward

* Dr. Douglas M. Gay, Wilmington.

the portal spaces around which the liver cells appear normal.

Kidney: Blood vessels are markedly congested. The epithelium of the convoluted tubules is swollen and the lumens of these tubules contain albuminous fluid.

Anatomical Diagnosis.

1. Rheumatic Heart Disease
   Tricuspid, mitral, and aortic stenosis
   Hypertrophied heart
   Pericarditis
2. Congestion of viscera, hydrothorax, ascites, and edema of lower extremities.
3. Pleural adhesions.

DISCUSSION

The clinical type of rheumatic fever illustrated in this case is the subacute polycyclic type, which has been well described by Griffith.[1] In this type of rheumatic fever the mortality is greatest. With repeated cycles of activity, the most extensive cardiac enlargement and the most frequent signs of endocarditis and pericarditis are found. During one or all of the cycles pneumonitis may occur. After the first cycle polyarthritis is usually absent, and the response to salicylate therapy is poor. Congestive heart failure complicating carditis occurs almost exclusively in this group of cases.[1]

Penicillin therapy for subacute bacterial endocarditis was instituted in this case in the absence of positive diagnostic criteria. The result of the penicillin treatment confirms previous observations that penicillin is not beneficial, and may be harmful in rheumatic fever except for intercurrent infections.[2]

Intravenous glucose administered as 25 cc. of 50% solution twice daily partially covered with 10 units of insulin plus an increase of the oxygen content of the inspired air to about 45 to 50% oxygen has been advocated in the treatment of selected patients with rheumatic carditis and congestive failure.[3] In this case, valvulitis and pericarditis were the predominant pathological changes, with the result that there was extensive mechanical cardiac disability in the presence of rheumatic activity.

Intravenous glucose and oxygen were not used in this case. However, in view of the mechanical disability of the heart it is doubtful that anything could have been accomplished by this form of therapy other than to prolong the patient's life for a short interval.

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RUPTURE OF THE UTERUS*

A Case Report with Review of Literature

OLIVER A. JAMES, M. D., and
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Milford, Del.

In February, 1947, we had one case of rupture of the uterus and because of the rarity of that complication we present the following:

It occurs most often during labor but may occur any time during the last trimester of pregnancy. The condition was first recognized and described by Guillemau, (1550-1613). The majority of cases are preventable, according to Drs. P. B. Bland and L. Goldstein. The incidence is highly variable, depending mostly on frequency of contracted pelvis. In Bucharest it was 1:300 deliveries; in Royal Maternity Hospital in London 1:6124; in Baltimore at Johns Hopkins 1:1010; at Boston City Lying In 1:1188; at New York Lying In 1:2260; University Hospital in Cleveland 1:2756; and in China 1:220.

The rupture in the majority of cases is of the complete type, meaning through the peritoneum. It may occur spontaneously and under this heading some authors, such as Dr. Bland include only those due to uterine abnormalities. More often it happens during some operative procedure, and this is termed traumatic. With the spontaneous cases some very interesting findings have been noted by Dr. Eastman and he considered them as contributory factors: (1) The average age rather high, above 36 years; (2) average parity, 6.4; (3) contraction of pelvis in 41.2% of the cases; (4) average weight of fetus 3946 grams (8 lbs., 11 oz.).

The most frequent causes are weak Caesarian scars, version and extraction deliveries, administration of oxytocics, and instrumental deliveries. Dr. Bland listed them anatomically: (1) those by the pelvis from contraction, malformation, or neoplasms; (2) by the uterus

*Read before the Monthly Staff Meeting, Milford Memorial Hospital, May 13, 1947.

from scars of prior operations, dry labors with retraction ring, acquired or congenital cervical stenosis, neoplasms, or pendulous abdomens with anteflexion of the uterus; (3) by the foteus from malposition and mal-presentation, overdevelopment of foetus, hydrocephalus, foetal neoplasms, multiple gestations, or monstrosities; and (4) those by external causes such as manual dilatation of cervix, version, instrumental delivery, manual extraction of placenta, administration of oxytocics and external violence of abdominal wall.

In regards to previous Caesarean sections, all writers advocate repeated sections in cases of contracted pelvis. Some including Bill, Barney, and Melody of Cleveland, and Tollefson of California urge "once a section always a section." As for the type, all reports show there are less ruptures in subsequent pregnancies with the low cervical. Holland reported 25% after classical type, with 3% after low cervical. At the Chicago Lying-In Hospital none in 1000 low cervical Caesarians; and Bill, Barney and Melody, of Cleveland, stated that all their ruptures followed classical. Yet they also say: "Always a section after the first." The repeated section should be done about two weeks prior to expected confinement.

All writers have put version and extraction deliveries very high on the list of causes. This is easily understood as there is a lot of increased tension and pressure on the uterus during the procedure.

As for oxytocics, nearly every report places them second to maternal and foetal disproportion. Dr. De Lee has been quoted to say that indiscriminate use of pituitrin is one of four major causes responsible for persistently high maternal and foetal death rates in the United States. He considered the use of pituitrin as a criminal act if it was given before delivery of child.

The most constant symptoms of the complete type are cessation of labor and generalized abdominal pain and tenderness. Frequently the patient feels a sudden tearing during a strong uterine contraction. Severe shock rapidly develops and continues unless effective treatment is soon started. Palpation of the abdomen gives a sensation of baby being just under the skin.

The most important thing is to recognize the symptoms and findings indicating impending rupture. Bland and Goldstein has listed them as: (1) prolonged labor with progressively stronger and more violent pains; (2) acceleration in maternal pulse rate from 80 per minute to 120-160, associated occasionally with an elevation in temperature; (3) ascent of contraction ring to a level between the pubis and navel at times approaching level of umbilicus; (4) general exquisite abdominal tenderness with marked tension of lower abdominal pain; (5) drawn and anxious facial expression showing an excessive degree of suffering; (6) swelling and discoloration of pinched vaginal portion of cervix; (7) gradual rise of foetal heart rate in intervals between uterine contraction indicating circulatory embarrassment as a result of excessive pressure from prolonged contractions.

The proper treatment is surgery as soon as the condition of patient will permit. Transfusions of whole blood are the best stimulant, but one should use any intravenous fluids until blood is available. In some cases the uterus has been repaired after delivery of the baby and placenta, but as a whole it is better to do a hysterectomy, as there is less chance for severe infection, and a subsequent pregnancy in a previously ruptured uterus is very dangerous.

The mortality rate for the mothers is very high—20% in some centers, but it depends entirely in how soon the condition is recognized. The foetal mortality is much higher—80% or more.

There are recorded some very unusual cases. Dr. Bland observed a rupture in a patient who had barely reached her sixth month. Two years prior she had been delivered by Caesarean section. Dr. B. H. Carroll, of Toledo, also observed a spontaneous rupture without warning, in a 22-year-old woman who was only eight months pregnant. She had had a low section at age 19 for placenta previa and a spontaneous delivery at age 21. Schwartz and Kurzrok, of Brooklyn, reported in 1936 a case in which the ureter had been ruptured. There are several instances of finding lacerations of the cervix extending up into the body of the uterus after apparently normal spontaneous deliveries, and also after application

of low forceps. In practically all of these there was record of pituitrin having been given to stimulate labor prior to delivery of foetus. Bleeding following delivery was the key to the situation, causing investigation of the cervix. Drs. Sinclair and Miller, of Whiteville, North Carolina, had a case with rupture following spontaneous delivery of first baby of a twin pregnancy. Previous uterine infection, myomectomy, plastic operations on the cervix have been contributing factors in some cases reported.

At the Milford Memorial Hospital there have been, since 1933 through 1946, over 3200 deliveries, with two cases of rupture of the uterus. Considering the number of complications that are referred to here, this is an excellent record of which the whole staff should be proud. The first case was in March, 1933, in a woman who had had four previous normal deliveries. Prior to her admission she had been given repeated small doses of pituitrin to accelerate labor. A Porro section was done, with delivery of a dead baby. She made an uneventful recovery.

The second case occurred in February, 1947, with a 38-year-old woman who had had two previous normal spontaneous deliveries. Her first labor in 1942 was only six hours long and the baby weighed 8¾ pounds. The second labor was in 1944 and only four hours in duration; that baby weighed 9¼ pounds. The third pregnancy had been uneventful until she went into labor at the end of term about 4 p. m., February 9, 1947. She progressed well with increasing contractions until about 1:00 a. m., when she was found to be almost fully dilated. Shortly afterward the pains ceased and remained so. At 7:00 a. m. she was in shock and complaining of general abdominal pain. After administration of intravenous plasma a laparotomy was done with extraction of a dead male foetus from the peritoneal cavity. The baby weighed more than 10 pounds. There was a long tear in the left side of the body of the uterus extending down through cervix into the vagina. A supravaginal hysterectomy was done. She had received no pituitrin or external stimulation at all during labor. Evidently the complication was due to over-development of the foetus. She recovered nicely. However, on the fifth post-operative day she developed signs of phlebothrombosis of the right leg and immediately a bilateral ligation of her superficial femoral veins was done. Her temperature rapidly subsided and returned to normal four days later. She was discharged on her eighteenth post-operative day. She was seen six weeks later, April 7, 1947, and was then able to do the greater part of her housework with no marked difficulty. Although she noted slight edema at night of her right ankle, there was no pitting edema at this examination.

In conclusion, the important points to remember are: (1) one should be alert to avoid a false sense of security with multiparas; (2) be quick to diagnose prior to onset of labor any disproportion, either due to minor contraction of pelvis or over-development of baby; (3) prevent rupture of old operative uterine scars; (4) avoid as much as possible version and extraction procedures; and (5) do not use oxytocics prior to delivery.

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+ Editorial +

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ONE HUNDRED YEARS OF ORGANIZED MEDICINE

The waves lashing the beach at Atlantic City with a million years of froth to their credit may laugh at the idea of a centennial celebration. But when we consider the progress of the past century as compared to the eons through which the Indians passed, singing, dancing, drumming, and rattling evil spirits away, the progress of American medicine has been phenomenal.

Even though Cabeza de Vaca, the shipwrecked Spaniard, initiated surgery on this continent in the present state of Texas with a sharp stone in the early part of the sixteenth century, there was little constructive progress before the American Medical Association was organized. The first American medical history was published in 1828. This was James Thacher's *American Medical Biography*. The next was by Stephen West Williams in 1845. This biographical material, dealing with the hardy, courageous physicians chiefly along the eastern seaboard, indicated that a solid foundation was being prepared for the American Medical Association, which was organized in 1847. It is to be hoped that when the modern medical great enter upon their centennial felicitations in Atlantic City this June, they will not forget the pioneer American physicians who went through "hell and high water" to give the profession stability of character and to prepare the way for scientific progress. Aside from the epoch-making discovery of general anesthesia, which was demonstrated at the Massachusetts General Hospital one year before the American Medical Association was founded, and aside from the control of yellow fever, American medicine has made no epochal advances but has contributed much toward national and international weal. It has given to the people of the United States the best medical service to be had anywhere in the world today.

The question arises, has the American Medical Association always made the most of its opportunities? Carried along with the last half century's scientific progress, the American Medical Association, in the opinion of the writer, has lost a great opportunity in the field of legitimate lay publicity. It was natural to be satisfied with the progress of scientific medicine and to assume public satisfaction. But the truth though crushed to earth is rising. The mass ignorance concerning the meaning of medical progress and its value to the individual and to society is appalling. In retrospect it is natural to wonder if the American Medical Association might not have more effectively educated the public as to the personal and general value of medicine as practiced in the United States today. The American Medical Association could have gone directly to the public with a sustained educational program and indirectly through the general practitioner who needed to be alerted and stimulated. Perhaps it is not too late. The next hundred years will tell.

Editorial, *J. Okla. S. M. A.*, June, 1947.

MISCELLANEOUS

The Problem of Specialty Boards

Speaking to the American Orthopedic Association in 1947, Dr. J. Albert Key in his presidential address voiced alarm because of the rigidity of the standards set up by the various specialty boards to determine the qualifications of the practitioners of medicine or surgery who wish to be admitted to the practice of their professions as qualified specialists. We quote from the standards set up by the American Board of Orthopedic Surgery:

1. One year rotating internship.
2. One year in adults' orthopedic surgery.
3. One year in children's orthopedic surgery.
4. One-half year internship in fractures.
5. One-half year full-time study of fundamental subjects: anatomy, pathology, bacteriology, physiology, and biochemistry.
6. Two years in practice limited to orthopedic surgery.

To those interested in the qualifying of specialists by boards we recommend the close reading of Dr. Key's address, which we cannot reprint here.[1]

We defy any millionaire aspirant—one to whom time and money are no object—to find the internships which are required by the American Board of Orthopedic Surgery. If he could, he would then be stymied by item No. 5; and after he had passed that, how could he serve two years in the practice of orthopedic surgery as required in item 6? He is still unqualified. How could the Board recommend that? According to them, he should be restrained from practicing as a specialist without their license, though qualified by the state or national boards to practice medicine and surgery.

There is thus created a dilemma. The specialty boards have assuredly done an excellent and praiseworthy job of elevating standards. Nobody would undo that work. But in all uplifting there is a point beyond which too many other values must be sacrificed to gain one objective. Has that point been reached?

Of course there must be qualifications set up for the fitness of those who exercise over the rest of us that power of life and death. Hitherto this has been done by the State Board Examinations and by the reputation—good or bad—which a surgeon acquired in his community. The judgment of his peers. Remember?

We feel that to allow self-styled, self-appointed specialists to crown themselves as censors to determine the qualifications of others to earn their living in a certain way can be dangerous.

Is there not a place in the practice of the profession of surgery for men to have drifted along in it and by some freak of fortune—war, for example—find themselves with an absorbing interest in orthopedic surgery, or plastic surgery, or any specialty for which their general experience and their later interest makes them qualified? The Board system takes us back to the medieval days of Henry VIII, when the surgeons, forced to unite with the barbers, set up the rigid apprenticeship and guild system, a partnership from which they were not able to escape until 1740.

Examiners on boards are rarely omniscient. May we remind the members of these boards, who are now so blithely setting the standards for their successors, that, brilliant men as they may be, outstanding as they are in the specialties, they are older men who never had to pass such examinations themselves and, if faced with them, could probably not do so now.

Our purpose in raising the question of present practices in qualifying doctors as specialists is not pointless criticism of the boards, but sincere evaluation of method, and some remarks on possible abuses. The boards have done too much good to risk having their work jeopardized by a senseless wave of reaction.

There must be a moderate course realistically related to human and economic values which could preserve the gains without sacrifice of abilities obtained through simple experience. We know of no way to improve matters except by raising the question for free discussion.

We realize that these associations, these boards, are set up out of zeal for the protection of the people. With the passage of the years many of them seem to have been in-

1. Key, J. Albert: J. Bone & Joint Surg. 29: 2, January, 1947.

vaded by the corruption of the self-interested. Those who are now in, being after all human beings, are potentially able to keep others out. They could reserve their privileges for their sons, their assistants—in short, for their successors. We fear a reversion to the guild system. We seem to remember that the "common man" did a pretty good job of protecting himself in the past. He simply stayed away from the incompetent practitioner and let him starve to death.

To those of our readers who are really interested in every aspect of this perplexing problem we recommend an article in the *Saturday Evening Post* of March 15, 1947 (Vol. 219, No. 37, page 23) by Comdr. W. J. Lederer, USN.

He got himself into Annapolis and subsequently attained his present rank without any formal training. He devoted sedulous and unflagging attention to the care of the *head*. It's a good idea.—Editorial, *N. Y. S. J. M.*, June 15, 1947.

Cash Sickness Benefits for Railroad Workers

A cash sickness benefit system for railroad workers began operating throughout the nation on July 1. These benefits were added under the 1946 amendments to the Railroad Unemployment Insurance Act and provide partial compensation for wage loss due to disability on the same basis as that due to unemployment.

All disabilities which prevent railroad employees from working, regardless of how or where they occur, are covered under the program.

A physician's statement of sickness will be required before claims can be paid. It is believed that the program will require about 65,000 medical examinations a year. Employees are free to choose their own doctors, and any physician to whom an employee goes for examination or treatment may supply the information required as initial proof of an employee's claim.

The forms on which medical information will be requested from a physician are the "Statement of Sickness" and the "Supplemental Doctor's Statement." The first mentioned form is intended primarily to obtain information at the beginning of each illness, and the second is intended to obtain additional information only when such information is needed later on in the same illness. The statements are designed to furnish as simply and as conveniently as possible for the physician, the minimum information required for Board purposes.

The "Statement of Sickness" on which the medical evidence is to be furnished must be mailed to the appropriate office of the Railroad Retirement Board within seven days after the first day claimed as a day of sickness, or the employee may lose part of his benefits. Claims for succeeding fourteen day periods may be allowed for a predetermined period as indicated by the medical evidence on the doctor's initial statement, but in continuing illnesses supplemental information about the patient's illness may also be requested from the physician.

Claims will be filed and adjudicated in the regional offices of the Railroad Retirement Board. These offices are located in Atlanta, New York, Cleveland, Chicago, Dallas, Kansas City, Minneapolis, Denver and San Francisco and serve the adjoining territories. Each will have a physician who will act as a medical consultant. Additional information about the program may be obtained from any of the aforementioned offices.

Too often it is assumed that the control of tuberculosis is solely the health department's domain of action. This is not true, nor can it ever be true, so long as men practice the ancient art of medicine. The family doctor in the city, the country doctor going about from farm to farm, the village doctor in his office over the drug store know the people, have their trust, and guide their physical destinies. The educational pamphlets of a hundred organizations cannot have the enduring effect nor the permeating persuasiveness of the doctor's personal word.—Herman E. Hilleboe, M. D., Pub. Health Rep., Dec. 6, 1946.

Good medical care is rarely cheap, and cheap medical care is rarely economical.—N. Y. Acad. Med. Rep. on Med. in Changing Order, Commonwealth Fund, 1947.

BOOK REVIEWS

Pharmacopoeia of the United States of America, 13th Revision (U. S. P. XIII). Pp. 957. Cloth. Price, $8.00. Easton, Pennsylvania: Mack Publishing Co., 1947.

This is the official opus of the U. S. Pharmacopoeial Convention and contains monographs on some 642 medicinals. It is the first of the "five year" revisions, the previous ones having been ten year revisions. Also, for the first time the English titles precede the Latin ones, with obvious advantages. It became official on April 1, 1947. The purchase price entitles the buyer to a copy of a bound supplement, should one become necessary. The U. S. P. is a sine qua non for all those who manufacture or prepare medicinals.

A History of the American Medical Association: 1847-1947. By Morris Fishbein, M. D., with the Biographies of the Presidents of the Association by Walter L. Bierring, M. D., and with Histories of the Publications, Councils, Bureaus, and Other Official Bodies. Pp. 1226. Illustrated. Cloth. Price, $10.00. Philadelphia: W. B. Saunders Company, 1947.

This book is published in recognition of the 100th Anniversary of the American Medical Association celebrated at the Centennial Session in Atlantic City, June 9-13, 1947. It is written for a general audience as well as for the medical profession. It reviews the rise of the Association as a great organization, and has reference particularly to the campaigns to raise the standard of medical education, to eliminate quackery, to improve industrial health, to eliminate nostrums and frauds in the medical field, and to provide a wide distribution of a high quality of medical care. All of these interests are of public as well as medical importance.

Delaware's only A. M. A. President (1867), Henry Ford Askew, is briefly sketched. His photograph is a new one to us, evidently much later than the one in "The Medical Society of Delaware: 1789-1939," by Dr. Meredith I. Samuel, of Wilmington.

Fishbein, Bierring and Company have done an outstandingly good job of writing, and that praise applies equally to the job of printing, done by the Saunders Company who, despite the late arrival of considerable copy (e. g., Dr. Askew's signature, which we personally know was difficult to locate) met their deadline just in time to present the first copy to President Bortz at the Centennial Convention. Naturally, in such a rush job, there are typographical errors, but surprisingly few; silliest is caption on p. 160, where Askew's successor, Samuel D. Gross (1805-1884), appears as President in 1898! In the text (p. 612) the year is given correctly as 1868. We blame the copy more than the compositor. So, congratulations to both companies.

Derived from carefully preserved official records, this history is really authoritative, and is recommended to all, physicians and laymen alike, who are interested in American medicine and American medical care. Especially recommended to Messrs. Wagner, Murray, Dingell, Pepper et al., for obvious reasons.

A Textbook of Medicine. Edited by Russell L. Cecil, M. D., Professor of Clinical Medicine, Cornell University Medical College; with assistance of Walsh McDermott, M. D., Associate Professor of Medicine, Cornell University Medical College. Associate Editor for Diseases of the Nervous System: Harold G. Wolff, M. D., Associate Professor of Neurology, Cornell University Medical College. Seventh Edition. Pp. 1730, with 244 illustrations. Cloth. Price, $10.00. Philadelphia: W. B. Saunders Company, 1947.

This new Cecil represents an increase over the sixth edition of 164 pages of text, and 48 illustrations. Because of war conditions 54 subjects had to be rewritten by new contributors. In addition, 16 new subjects are included. Several sections have been rearranged. The list of contributors totals 162, selected especially because of their knowledge of this particular subject.

The format shows a slightly larger page, and retains the double columns which proved so popular in the previous edition, as well as the tables of normal values for clinical examinations immediately preceding the index.

Cecil is still one of the leading texts of American medicine. We have the impression we see more quotations from it than from any other. This seventh edition will be much quoted too.

Diseases of Metabolism—Detailed Methods of Diagnosis and Treatment—A Text For The Practitioner. Edited by Garfield G. Duncan, M. D., Director of Medical Division, Pennsylvania Hospital; Clinical Professor of Medicine, Jefferson Medical College, Philadelphia. Second edition. Pp. 1045, with 167 figures, 8 in colors. Cloth. Price $12.00. Philadelphia: W. B. Saunders Company, 1947.

The title of this book eminently befits its contents. Covering all the material usually found in such a work, it comes over, logically, into the realms of nutrition, endocrinology, and hematology. New chapters covering the thyroid gland and the kidneys have been added. Each chapter has been brought up to date, the latest researches being included, except those that have little or no practical application at the present time. Controversial subjects have been largely omitted. An appendix includes tables of composition of foods, height and weight, and basal energy requirements.

With one exception, ample space has been given to each subject. We think the chapter on Undernutrition, meaty as it is, might have been amplified considerably. We find the patients complaining about being "too skinny" outnumber those bemoaning their adiposity: these latter folk generally do not bemoan at all—they laugh at it.

We heartily recommend the book.

Surgical Pathology. By William Boyd, M. D., Professor of Pathology, the University of Toronto, Canada. Sixth edition. Pp. 858, with 530 illustrations, including 22 color figures. Cloth. Price, $10.00. Philadelphia: W. B. Saunders Company, 1947.

Boyd's object was to present those aspects of pathology which would prove useful to the surgeon. That he succeeded is evidenced by the appearance of a sixth edition. His text correlates the living pathology of the operating room with the basic dead pathology of the autopsy room, and abounds in practical clinical points of diagnosis that often point the way to proper treatment. The style is quite readable—compact yet not terse, good example, the two pages on Peritoneal Adhesions.

An entirely new section has been added dealing with the pathology of congenital heart disease. Other new material includes tumors of the larynx, pinealoma, Bittner's milk factor in relation to breast carcinoma, avitaininosis in mouth cancer. The Papanicolaou vaginal smear in cervical cancer, nodules dysplasia of bone, inflammatory nodules of muscle in chronic arthritis, and fibrositis of the back.

This actually is a Pathology for the surgeon; it needs no commendation from us.

Diseases of the Chest: With Emphasis on Xray Diagnosis. By Eli H. Rubin, M. D., Attending Physician, Division of Pulmonary Diseases, Montefiore Hospital and Country Sanatorium, New York; Visiting Physician in Tuberculosis and Physician-in-charge, Chest, Clinic, Morrisania City Hospital, New York. Pp. 685, with 355 illustrations, 24 in color. Cloth. Price, $12.00. Philadelphia: W. B. Saunders Company, 1947.

The complete description of any one of the described diseases, with its etiology, pathology, xray study, medical and surgical phases, treatment might well consume an entire book. The author, however, has merely sketched the commoner diseases, as well as some of the more frequent neoplasms.

While the work contains many excellent radiographic reproductions, one is impressed by the text, which indicates the relative ease and reliance upon the fluoroscopic study alone. This reviewer, however, is of the opinion that physicians planning the study of chest diseases may be misled by the brevity and implied ease with which sufficient information may be acquired.

The book impresses us as an excellent "quiz compend."

Harofe Haivri

The Spring issue of Harofe Haivri (the Hebrew Medical Journal), a semiannual bilingual publication, contains, in the medical section, the articles on the clinical use of streptomycin, anticoagulant therapy in the treatment of thrombosis and narcoanalysis and narcosynthesis. The section on Palestine and Health contains an enlightening statistical survey on the health of the young generation of Palestine, and a paper on the historical and archaeological aspects of the hot springs of Tiberias.

Under the heading of historical medicine there is a treatise on medical ethics by Asaph the physician, who lived in the 9th century.

The original articles are summarized in English to make them available to those who are unable to read Hebrew.

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